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The role of nitric oxide in the regulation of neurotransmitter release and processes of exo- and endocytosis of synaptic vesicles in mouse motor nerve endings

Yakovleva O., Shafigullin M., Sitdikova G.
Kazan Federal University, 420008, Kremlevskaya 18, Kazan, Russia

Abstract

In experiments with mouse diaphragm muscle, the effects of a nitric oxide (NO) donor, S-nitroso- N-acetyl-DL-penicillamine (SNAP), and an NO-synthase blocker, NG-nitro-L-arginine methyl ester (LNAME), on transmitter release and processes of exo- and endocytosis of synaptic vesicles in the motornerve ending were studied using electrophysiological and fluorescence techniques. During single stimulation of the motor nerve, SNAP reduced and LNAME did not change the amplitude of the endplate currents and both of the drugs did not affect spontaneous transmitter release. During high-frequency stimulation (20 Hz, 3 min) SNAP increased and LNAME slowed the depression of the amplitudes of endplate potentials (EPPs) compared to the dynamics of EPPs in the control. In experiments using the fluorescent dye FM 1-43, it was shown that the NO donor induced a decrease and LNAME induced an increase in the fluorescence intensity of motor-nerve endings loaded with dye during stimulation at a frequency of 20 Hz for 30 s compared to the control. At the same time, the rate of dye unloading from the terminals that were preloaded with FM 1-43 was higher after preliminary application of SNAP and lower after preliminary application of LNAME. It was suggested that exogenous and endogenous NO in the mouse neuromuscular synapse caused the depression of neurotransmitter release as a result of the suppression of synaptic-vesicle recycling due to a decrease in endocytosis or/and mobilization of synaptic vesicles from a recycling pool to the exocytosis sites. © 2013 Pleiades Publishing, Ltd.

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Keywords

exo- and endocytosis, FM 1-43, motor-nerve ending, neurotransmitter release, nitric oxide, synaptic vesicles